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ABSTRACT

The two-part paper examines the neurobiological processes of synapse overproduction, synapse elimination, and issues of language acquisition and attrition. The first part consists of diagrams and notes explaining some basic terms and concepts of neurobiology: cortex; white matter; neuron; synapse; synaptogenesis; and development and organization of neuronal connections. The second part details, in narrative, two processes correlated with maturation (synaptic elimination and stabilization of the synapses that survive the elimination stage) and their implications for second language learning and first language attrition. These processes are then applied to hypothetical cases of children emigrating from their first-language environments before the age of 8 and after the age of 12 to explain patterns of language acquisition, maintenance, and loss. (MSE)

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A Neurobiological Basis for SLA and First Language Attrition

Part I. Stella de Bode

The nervous system - some basic terms and concepts

Cortex = gray matter - approximately 10 billion nerve cells linked to one another via hundreds of thousands of miles of connections. Only 4-6mm wide

White matter - the axons

I. Neuron - basic information processing unit of the nervous system

Each neuron has cell body and branches (off the body of the cell). There are two types of branches: 1. **axon** -

-conducts impulses away from cell body

-transmits nerve impulses from one neuron to the next

-can reach a yard or more

2. **dendrite**-

-receive impulses and contacts from other neurons

II. Synapse

Synaptic junction - site of communication and contact of two neurons

-usually there is no physical contact of parts of cells

-contact is chemical by means of neurotransmitter

Fig. 1 Pyramidal cell

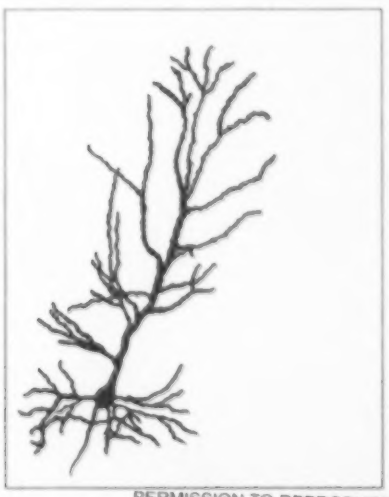
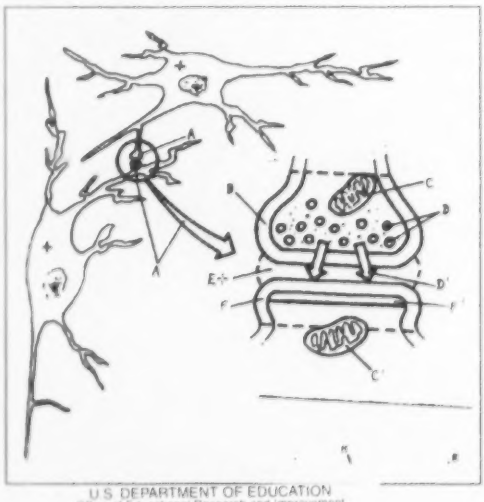


Fig. 2 Synapse structure



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III. Synaptogenesis = synaptic development

-as soon as a developing axon reaches its target neuron a synapse is formed

-synaptic proliferation occurs in different areas of cortex at the same time

-the completion of synaptogenesis is the hallmark of functional maturity

-synaptic density might be related to functional capacity

-synapses form prenatally but do not reach adult numbers until well after birth

-maturational pattern involves overproduction followed by consolidation until numbers reduced to adult levels

-consolidation (though not so profound) may continue into puberty

IV. Development and organization of neuronal connections

1. Establishing connections between neurons is the cardinal achievement of neurological development

2. The nature of cell connectivity is a crucial aspect of the neurological underpinnings of cognition, including language

3. Neurons in many areas of cortex appear to be grouped into regularly spaced vertical columns, resulting in modular organization of cells throughout cortex

4. Exceptional functional plasticity in childhood might be the result of excess of available synaptic connections. These "extra" connections are being eliminated by puberty and after that creating new synaptic pathways (which is a substrate of any learning) is the result of hard work.

Fig. 3 Synaptic density and total synapses in VC as a function of age

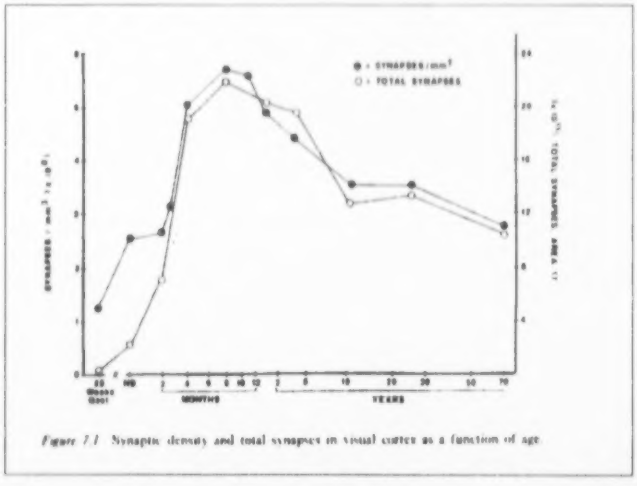

Figure 7.1 Synaptic density and total synapses in visual cortex as a function of age.

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A Neurobiological Basis for SLA and First Language Attrition

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Part II

This paper explores connections between neurobiological processes of synapse overproduction and synapse elimination and issues of language acquisiton/attrition. Neurobiological support for language attrition is less investigated than sociolinguistic or linguistic aspects but research in this area offers interesting insights. It is possible that language loss similar to language acquisition is the result of neural plasticity (Yoshitomi, 1992). Neural plasticity allows input to alter the configuration of existing knowledge networks in memory storage. Vulnerability to attrition is greatest with respect to recently acquired, unconsolidated knowledge. Information that survives competition and reorganization becomes less prone to erosion processes.

The support for neurobiological model proposed by this paper was drawn from the theory of synapses overproduction and selective stabilization of developing synapses (Huttenlocher, 1994; Changeux & Danchin, 1976). The majority of the research in this area was conducted on the primary visual cortex of human and non-humans with data for other areas of frontal cortex much less complete. However, some results and observations of the visual cortex study can still be extended to other areas of the cortex on the basis of the hypothesis offered by Rakic et al (1986). In this pioneering study, it was argued that there exists the concurrent overproduction of synapses in diverse regions of the primate cerebral cortex. Not only does this theory contradict the traditional view of the hierarchical development of cortical regions by presenting the data of simultaneous synaptogenesis in all areas and regions of cortex, but it also suggests that the maturation of the nervous system coincides with synaptic elimination. In other words, maturation occurs when the synaptic number is altered on the basis of experience by causing the selective survival of certain

synapses. So, there are two processes correlated with maturation: synaptic elimination and stabilization of those synapses that survived the elimination stage. Let us consider both processes and their functional correlates, including conditions that underlie their existence.

I started with an examination of the major reason for synaptic overproduction and elimination. What is the neurochemical mechanism of synaptic overproduction and what causes their consequent elimination? It has been proposed that neurons in many regions of the developing nervous system are overproduced initially and appear to compete for survival (Purves & Lichtman, 1980). A major reason for these processes is to regulate the number and distribution of synapses made within a target cell, i.e. initial redundancy and subsequent elimination ensure innervation of all cells and, later, a quantitatively appropriate distribution of synapses among the target cells. The underlying mechanism of this competition for survival remains unclear, but it seems that the number of neurons in an innervating population is defined by feedback and consequent adjustment. Purves and Lichtman describe this process in terms of each axon progressively confining an increasing number of synaptic contacts to a smaller number of target cells where positive feedback is induced by uptake of trophic material, i.e. one of the terminals begins to gain a larger share of the trophic agent than its competitors which leads to its consequent stabilization. Changeux and Danchin (1973), in turn, argue that the notion of the "trophic agent" is not relevant to the discussion and stabilization is achieved by means of neurotransmitters, namely, an anterograde factor and a retrograde factor which govern the transition from unspecified circuits to specified ones. Though this question remains open it should not prevent us from considering functional correlates of synaptic overproduction.

From the large body of experimental data from animal models it is feasible to suggest that elimination of "redundant" connections is an important part of the nervous system's maturational process. Moreover, elimination depends on the amount of electrical activity in the developing system, i.e. functional demands and conditions during a critical period, when

modifications are easily achieved at the expense of transient fibers which might be incorporated into a permanent system (Huttenlocher, 1994). Information about synaptogenesis and synaptic elimination in humans is not as complete as that for animals. However, some data, especially those for the visual cortex are still available. The approximate total number of synapses in the visual cortex at birth was found to be only about one-sixth that in the adult (Huttenlocher, 1994). Between birth and age 6 months, there is a rapid growth of synaptic connections resulting in a number that is considerably larger than that of the adult. This might be considered evidence of synaptic overproduction in the cerebral cortex and leads to the next stage of its development, synapse elimination. Synapse elimination in visual cortex has been found to begin at about at about age 1 and is complete by about age 10. The time line for the frontal cortex is somewhat slower, with maximum synaptic density reached by the age of 1 year and synapse elimination occurring after the age of 7 until puberty. Huttenlocher (1982) found that up to 42% of synapses in the visual cortex are eliminated with the increasing complexity of a neural system. The period of synapse elimination in the visual cortex is proposed by Wilson (1988) to be a period of visual "functional fine-tuning, under environmental control." Based on this critical period under which visual development can be altered and this period roughly coincides with the onset and the completion of synapse elimination, Huttenlocher (1994) concluded that exceptional *functional plasticity to afferent input is because of "excess of available synaptic connections"*. Despite the lack of hard-core data I speculate that a similar process may occur in other brain regions including those related to language. With the process of elimination fully completed it is possible to determine why functional plasticity is lost in adults.

It is important to consider the conditions under which some synapses are eliminated while others are getting stabilized. Electric activity in the developing neural circuits seems to be crucial in establishing neural connections. Moreover, synapses are eliminated more rapidly under the conditions of increased activity, thus, achieving more effective "fine-tuned" connections. Purves and Lichtman go even further suggesting that not only the level of

activity per se but an imbalance of activity among competing inputs is important. This is, synchronous activity might impede competition, but if it is persistent it is possible that instead of a single input remaining, synchronously active inputs will remain and become stabilized. In short, there is a critical period in the development of a nerve terminal when it requires a certain amount of activity to become stable. If conditions are favorable, then a labile and diffusible receptor becomes stable and resistant by stabilization reaction which is final and not reversible (Changeux & Danchin, 1973).

Before proceeding to a discussion of language attrition it is interesting to consider glucose utilization by the human brain. Quantative analyses of ICMRGlc shows that the brain follows a protracted glucose metabolic maturational course, i.e. the period of increased energy demand correlates with the functional maturation of the brain (Chugani, 1994). Chugani argues that the ascending portion of rapid ICMRGlc increase corresponds to the period of synapses overproduction, on the other hand, the slow decline of ICMRGlc roughly coincides with the phase of synapse elimination and marks the threshold when brain plasticity considerably diminishes.

The neurobiological model proposed here embraces the studies discussed above and provides a biological basis for a model of language attrition and SLA.

Incorporating the issues presented above in the literature review with the fact that dendritic growth rapidly slows down after about the age of 8 to 12, I made an imaginary division and, based on my experience with children losing their first language, divided them into two groups: children who have emigrated before the age of 8 and children who have emigrated after the age of 12. Taking into consideration the fact that the former group easily achieves near-native proficiency in the L2 and retains very little of L1 it is feasible to suggest that second language acquisition is achieved because of the availability of extra circuits that have not yet been eliminated. On the other hand, the first language deteriorates profoundly because stabilization of L1 circuits has not yet occurred and by the time it occurs L1 circuits must compete with L2 circuits against a background of increasing L2 input. As a result of

impoverished L1 input, synapses are eliminated at a slower rate and "fine-tuning" of L1 connections never happens. For the group of children who have emigrated after the age of 12, the situation is different. Only few if any extra circuits are available and in order to build L2 competence children utilize those circuits available to them, that is, L1 circuits. Second language acquisition after this point varies but, generally, it is often not complete. This might be explained by the fact that learners in their SLA rely heavily upon their already established connections and draw from their knowledge of L1. Language loss also varies but its degree cannot be compared to that of a younger group, i.e. the first language is less prone to total dissolution, maybe because of the continuous use of the same circuits.

In any case, the proposed model provides an explanation for language acquisition and language loss as consequences of two inverse processes characterizing neural plasticity (Yoshitomi, 1992): synapse overproduction and synapse elimination.